

Table 5.14 Exposure from incidental ingestion of water while swimming

Contaminant	2.5 year old child mg/kg-day	Adult mg/kg-day
Ammonia	NA	NA
Antimony	3.05e-09	1.92e-10
Arsenic	2.16e-10	1.36e-11
Barium	5.87e-09	3.69e-10
Beryllium	1.19e-11	7.46e-13
Cadmium	1.29e-10	8.13e-12
Chromium VI	1.49e-09	9.36e-11
Chromium (total)	9.39e-09	5.90e-10
Copper	5.90e-09	3.71e-10
Lead	5.46e-09	3.43e-10
Manganese	1.88e-08	1.18e-09
Mercury	1.34e-09	8.45e-11
Nickel	2.49e-08	1.56e-09
Selenium	1.20e-10	7.57e-12
Silver	2.19e-10	1.38e-11
Thallium	2.82e-11	1.77e-12
Vanadium	3.30e-10	2.08e-11
Zinc	3.30e-08	2.07e-09
Alkyl pyridine	NA	NA
2-Cyanopyridine	NA	NA
3-Cyanopyridine	NA	NA
Benzene	NA	NA
Ethanol	NA	NA
Isopropanol	NA	NA
Methanol	NA	NA
2-Picoline	NA	NA
3-Picoline	NA	NA
Pyridine	NA	NA
Toluene	NA	NA
NA = Not applicable		

Table 5.15 Exposure from dermal absorption while swimming

Contaminant	2.5 year old child mg/kg-day	Adult mg/kg-day
Ammonia	NA	NA
Antimony	2.94e-10	5.95e-11
Arsenic	2.08e-11	4.21e-12
Barium	5.67e-10	1.15e-10
Beryllium	1.14e-12	2.31e-13
Cadmium	1.25e-11	2.52e-12
Chromium VI	1.44e-10	2.91e-11
Chromium (total)	9.06e-10	1.83e-10
Copper	5.69e-10	1.15e-10
Lead	5.27e-10	1.07e-10
Manganese	1.82e-09	3.67e-10
Mercury	1.30e-10	2.62e-11
Nickel	2.40e-09	4.85e-10
Selenium	1.16e-11	2.35e-12
Silver	2.11e-11	4.27e-12
Thallium	2.72e-12	5.49e-13
Vanadium	3.19e-11	6.44e-12
Zinc	3.18e-09	6.43e-10
Alkyl pyridine	NA	NA
2-Cyanopyridine	NA	NA
3-Cyanopyridine	NA	NA
Benzene	NA	NA
Ethanol	NA	NA
Isopropanol	NA	NA
Methanol	NA	NA
2-Picoline	NA	NA
3-Picoline	NA	NA
Pyridine	NA	NA
Toluene	NA	NA
NA = Not applicable		

6 Dose-response characterization

In the previous section, we modeled potentially significant pathways of exposure, generating conservative estimates of the doses of contaminants that the MEIs may receive from emissions. In this section, we describe the toxicologic factors that the determination whether such doses constitute significant risk of harm.

By convention, the various adverse effects on health are grouped into two broad categories: cancer, and all other adverse effects.

6.1 Estimates of cancer potency and unit risk

Seven of the chemicals of potential concern are known to be carcinogens, either in rodents or humans or both (see Table 6.2). Each was assigned an estimated carcinogenic potency by the U.S. EPA, by the New York State Department of Health (NYSDOH), or, in the case of ethanol, by Cambridge Environmental Inc. (see Table 6.2, footnote d). The potency estimates derive either from the results of chronic bioassays in rodents or, less often, from the results of epidemiologic studies.

The dose-response factors, also termed "potency slope factors" or "unit risks," are used to derive quantitative estimates of carcinogenic risk for each chemical. Potency slope factors and unit risks are expressed on a risk per unit exposure basis. For oral and dermal doses, the potency slope factor P is expressed in units of kg-day/mg; the numerical value of P for a particular chemical corresponds to the estimated incremental risk of cancer conferred by an exposure of 1 mg/kg-d for life. Generally, estimates of P are derived for oral exposures, which we distinguish by the use of the subscript "o". Since P_o are usually derived for applied exposures (*i.e.*, they are based on the total intake of the contaminant), the extrapolation of the oral potency factor to estimate dermal potency factors must account for possible differences in the absorption of the chemical across the gut and as opposed to across the skin. We therefore estimate dermal potency factors for exposures to water (P_{dw}) by adjusting each oral potency factor (P_o) by an oral absorption coefficient (ζ_o) for that chemical, as shown below. Since exposures *via* dermal absorption from water are already calculated as absorbed doses (Section 5.12), no adjustment by an absorption coefficient through the skin is needed. Oral absorption factors are listed in Table 6.1.

$$P_{dw} = \frac{P_o}{\zeta_o} \quad (6.1)$$

Incremental cancer risks for inhalation are quantified by unit risk values (U_i). Expressed in units of $\text{m}^3/\mu\text{g}$, the numerical value of U_i for a particular chemical is interpreted as an estimate of the excess risk of cancer incurred from a lifetime of exposure to $1 \mu\text{g}/\text{m}^3$ of that chemical in the air.

The potencies and unit risks used herein are listed in Table 6.2. The notations "ND" — not determined — or "NA" — not available — apply to those chemicals that are not known or suspected to be carcinogens.

6.2 Estimates of dose-response for effects other than cancer

For end points other than cancer, we compare the doses estimated to derive from the NEPERA incinerator with three measures of acceptable doses, depending on whether the dose is oral, dermal, or inhaled. For oral and dermal doses, we identify or calculate a quantity called a reference dose (R_{ID}). An R_{ID} is an estimate of the daily dose of a chemical to which people (including sensitive subgroups) can be exposed for their entire lifetimes and not incur appreciable risk of harm. For inhalation exposure, a reference concentration (R_{IC} , in mg/m^3) represents an airborne concentration of a chemical that may be tolerated for a lifetime without causing (non-carcinogenic) adverse effects on health. If doses or concentrations projected to result from operations of the NEPERA incinerator are less than the appropriate reference doses or concentrations, no significant risk of harm is expected.

U.S. EPA and other agencies have estimated the R_{ID} for oral exposure to various chemicals, which we distinguish with a superscript "o" (R_{ID}^o); these values may be used to calculate the corresponding R_{ID} for dermal exposures to contaminants in water (designated R_{ID}^{dw}). For each chemical, we have calculated an R_{ID}^{dw} by adjusting the R_{ID}^o by the oral absorption factor ζ_o . Oral absorption factors are given in Table 6.1.

$$R_{ID}^{dw} = R_{ID}^o \zeta_o \quad (6.2)$$

NYSDOH has developed reference concentrations for several of the contaminants of concern in this risk assessment; for other contaminants, R_{IC} s are taken or derived from U.S. EPA assessments.

Lead is not evaluated for non-cancer health effects in this way. It is not currently known whether there is a threshold in the dose-response relationship for lead and its most important adverse effect — which is its apparent harm to the developing nervous system of the fetus or child. It is thus as yet undetermined whether there is some small "safe" dose for lead, or whether, in the alternative, any exposure to lead, however small, carries with it some non-zero risk of harm. Given this uncertainty, it is inappropriate to use a reference dose or reference concentration as a

means of evaluating the effects of various incremental exposures to lead. Instead, for this assessment, we evaluate the degree to which lead exposure increases concentrations in blood lead in the child MEI. The results are shown in Table 7.3.

Reference doses and concentrations used in this section are presented in Table 6.3.

Table 6.1 Oral absorption factors

Contaminants	Fraction of compound absorbed when ingested	Comments
Ammonia	NA	
Antimony	0.01	ATSDR, 1992a (b)
Arsenic	0.95	ATSDR, 1993a (a)
Barium	0.05	ATSDR, 1992 (a)
Beryllium	0.01	ATSDR, 1993b (c)
Cadmium	0.05	ATSDR, 1993c (a)
Chromium VI	0.02	ATSDR, 1993d (a)
Chromium (total)	0.02	ATSDR, 1993d (a)
Copper	0.6	ATSDR, 1990a (d)
Lead	0.5	ATSDR, 1993e (e)
Manganese	0.05	ATSDR, 1992c (a)
Mercury	0.15	ATSDR, 1992d (f)
Nickel	0.2	ATSDR, 1993f (a)
Selenium	0.8	ATSDR, 1989a (g)
Silver	0.21	ATSDR, 1990b (a)
Thallium	1	ATSDR, 1992e (a)
Vanadium	0.01	ATSDR, 1992f (a)
Zinc	0.3	ATSDR, 1992g (a)
Alkyl pyridine	NA	
2-Cyanopyridine	NA	
3-Cyanopyridine	NA	
Benzene	NA	
Ethanol	NA	
Isopropanol	NA	
Methanol	NA	
2-Picoline	NA	
3-Picoline	NA	
Pyridine	NA	
Toluene	NA	

Table 6.1 Oral absorption factors

- NA = Not applicable — no routes of dermal exposure are considered for the organics or ammonia.
- a - Based on the ATSDR Toxicological Profile for this compound. If quantitative information is lacking, an approximation is made. When the phrase "readily absorbed" is used, 100% absorption is assumed. When the phrase "limited absorption" is used, 1% absorption is assumed.
- b - The International Committee on Radiation Protection recommends a value of 1% for absorption of most forms of antimony (ATSDR, 1992a).
- c - Since beryllium and its compounds are poorly absorbed from the gastrointestinal tract. It is unlikely that beryllium is absorbed from the skin (ATSDR, 1993b). A default value of 1% is used.
- d - Value is fraction absorbed by humans; range was 15-97% (ATSDR, 1990a).
- e - Value is absorption fraction for dietary lead for children; only 15% of dietary lead is absorbed in adults (ATSDR, 1993e).
- f - Absorption fractions for inorganic mercury. Absorption fraction for metallic and organic mercury are 0.0001 and 0.95, respectively (ATSDR, 1992d).
- g - Absorption following oral exposure depends on the physical and chemical states of the compound and the dose regimen.

Table 6.2 Potency values for carcinogenic compounds of concern

Contaminant	CAS Number	Weight of evidence (f)	Oral slope factor		Inhalation unit risk		Dermal slope factor (kg-d/mg)
			kg-d/mg	Source	(m ³ /μg)	Source	
Ammonia	7664-41-7	ND	NA		NA		NA
Antimony	7440-36-0	ND	NA		NA		NA
Arsenic	7440-38-2	A	1.75e+00	IRIS, 3/94 (a)	4.30e-03	IRIS, 3/94	1.84e+00
Barium	7440-39-3	ND	NA		NA		NA
Beryllium	7440-41-7	B2	4.30e+00	IRIS, 3/94	2.40e-03	IRIS, 3/94	4.30e+02
Cadmium	7440-43-9	B1	ND		2.00e-03	NYSDOH, 1990a (b)	NA
Chromium VI	7440-47-3	A	ND		5.00e-02	NYSDOH, 1990b (b)	NA
Chromium (total)	7440-47-3	ND	NA		NA		NA
Copper	7440-50-8	D	NA		NA		NA
Lead	7439-92-1	B2	ND		ND		NA
Manganese	7439-96-5	D	NA		NA		NA
Mercury	7439-97-6	D	NA		NA		NA
Nickel	7440-02-0	A	ND		2.40e-04	IRIS, 3/94 (c)	NA
Selenium	7782-49-2	D	NA		NA		NA
Silver	7440-22-4	D	NA		NA		NA
Thallium	7440-28-0	D	NA		NA		NA
Vanadium	7440-62-2	ND	NA		NA		NA
Zinc	7440-66-6	D	NA		NA		NA
Alkyl pyridine		ND	NA		NA		NA
2-Cyanopyridine	100-70-9	ND	NA		NA		NA
3-Cyanopyridine	100-54-9	ND	NA		NA		NA
Benzene	71-43-2	A	2.90e-02	IRIS, 6/94	8.30e-06	IRIS, 6/94	NA
Ethanol	64-17-5		1.30e-04	(d)	3.70e-08	(e)	NA
Isopropanol	67-63-0	ND	NA		NA		NA

Table 6.2 Potency values for carcinogenic compounds of concern

Contaminant	CAS Number	Weight of evidence (f)	Oral slope factor		Inhalation unit risk		Dermal slope factor (kg-d/mg)
			kg-d/mg	Source	(m ³ /μg)	Source	
Methanol	67-56-1	ND	NA		NA		NA
2-Picoline	109-06-8	ND	NA		NA		NA
3-Picoline	108-99-6	ND	NA		NA		NA
Pyridine	110-86-1	ND	NA		NA		NA
Toluene	108-88-3	D	NA		NA		NA

Notes:

ND = Not determined; NA = Not applicable.

a - Converted from the unit risk for arsenic in drinking water (5e-5 L/μg) assuming a body weight of 70 kg and a drinking water ingestion rate of 2 L/day.

b - Recommended in the NYSDOH Ambient Air Criteria Document for this compound.

c - Based on the inhalation unit risk reported for nickel refinery dust.

d - This potency is derived by Cambridge Environmental Inc. using the single long-term bioassay of ethanol that showed a positive response (Radike, 1981). It corresponds to the best estimate of potency in Sprague-Dawley rats for hyperplastic liver nodules (for pituitary tumors, the value would be 1.2×10^{-4} kg-d/mg), extrapolated to humans by assuming equal response for equal doses on a bodyweight basis. The corresponding lifetime risk estimate for the average U.S. consumption of alcohol (approximately 139 mg/kg-d) is 1.8%. Doll and Peto (1981) estimate that alcohol is involved in the causation of 4% of all human cancers, corresponding to approximately 1% lifetime risk.

e - Extrapolated from the oral reference dose assuming an inhalation rate of 20 m³/day and a body weight of 70 kg.

f - U.S. EPA Classifications scheme as cited in IRIS, 3/94. A = known human carcinogen, B = probable human carcinogen, C = possible human carcinogen, D = not classifiable as to human carcinogenicity, and E = not carcinogenic to humans.

Table 6.3 Reference doses and reference concentrations for chemicals of concern

Contaminants	CAS Number	Oral RfD		Inhalation RfC		Dermal RfD (mg/kg-d)
		(mg/kg-d)	Source	(mg/m ³)	Source	
Ammonia	7664-41-7	9.71e-01	HEAST, 1993 (a)	1.00e-01	IRIS, 3/94	NA
Antimony	7440-36-0	4.00e-04	IRIS, 3/94	2.15e-02	(b)	4.00e-06
Arsenic	7440-38-2	3.00e-04	IRIS, 3/94	5.00e-04	(c)	2.85e-04
Barium	7440-39-3	7.00e-02	IRIS, 6/94	5.00e-04	HEAST, 1993	3.50e-03
Beryllium	7440-41-7	5.00e-03	IRIS, 3/94	1.00e-05	(d)	5.00e-05
Cadmium	7440-43-9	5.00e-04	IRIS, 3/94 (e)	2.00e-05	NYSDOH, 1990a (f)	2.50e-05
Chromium VI	7440-47-3	5.00e-03	IRIS, 3/94	1.00e-04	NYSDOH, 1990b (f)	1.00e-04
Chromium (total)	7440-47-3	1.00e+00	IRIS, 3/94	1.00e-04	NYSDOH, 1990b (f)	2.00e-02
Copper	7440-50-8	3.70e-02	HEAST, 1993 (g)	6.00e-04	(h)	2.22e-02
Lead	7439-92-1	NA	(i)	NA	(i)	NA
Manganese	7439-96-5	5.00e-03	IRIS, 3/94 (j)	3.00e-04	NYSDOH, 1989a (f)	2.50e-04
Mercury	7439-97-6	3.00e-04	HEAST, 1993	3.00e-04	HEAST, 1993	4.50e-05
Nickel	7440-02-0	2.00e-02	IRIS, 3/94	2.00e-05	NYSDOH, 1989b (f)	4.00e-03
Selenium	7782-49-2	5.00e-03	IRIS, 3/94	1.75e-02	(k)	4.00e-03
Silver	7440-22-4	5.00e-03	IRIS, 6/94	4.00e-03	(l)	1.05e-03
Thallium	7440-28-0	8.00e-05	IRIS, 6/94	2.20e-03	(m)	8.00e-05
Vanadium	7440-62-2	7.00e-03	HEAST, 1993	2.00e-04	NYSDOH, 1990c (f)	7.00e-05
Zinc	7440-66-6	3.00e-01	IRIS, 3/94	5.00e-02	NYSDOH, 1988 (f,n)	9.00e-02
Alkyl pyridine		1.00e-03	(o)	3.50e-03	(o)	NA
2-Cyanopyridine	100-70-9	1.00e-03	(o)	3.50e-03	(o)	NA
3-Cyanopyridine	100-54-9	1.00e-03	(o)	3.50e-03	(o)	NA
Benzene	71-43-2	4.70e-01	(p)	1.60e-01	(q)	NA
Ethanol	64-17-5	1.30e+01	(p)	4.50e+01	(r)	NA
Isopropanol	67-63-0	7.00e+00	(p)	2.30e+01	(r)	NA
Methanol	67-56-1	5.00e-01	IRIS, 6/94	1.75e+00	(k)	NA

Table 6.3 Reference doses and reference concentrations for chemicals of concern

Contaminants	CAS Number	Oral RfD		Inhalation RfC		Dermal RfD (mg/kg-d)
		(mg/kg-d)	Source	(mg/m ³)	Source	
2-Picoline	109-06-8	1.00e-03	(o)	3.50e-03	(o)	NA
3-Picoline	108-99-6	1.00e-03	(o)	3.50e-03	(o)	NA
Pyridine	110-86-1	1.00e-03	IRIS, 6/94	3.50e-03	(k)	NA
Toluene	108-88-3	2.00e-01	IRIS, 6/94	4.00e-01	IRIS, 6/94	NA

Notes:

ND = Not determined; NA = Not applicable.

a - Converted from the drinking water standard for ammonia of 34 mg/L assuming a body weight of 70 kg and a drinking water rate of 2 L/day.

b - Extrapolated from the lowest LOAEL listed in ATSDR (1992a) - for adverse cardiovascular effects in exposed workers - by applying a safety factor of 100.

c - Extrapolated from the lowest LOAEL listed in ATSDR (1993a) - for adverse cardiovascular effects in exposed workers - by applying a safety factor of 100.

d - Based on the National Emission Standards for Hazardous Air Pollutants (NESHAPS) 30-day standard of 0.01 µg/m³ as reported in NYSDEC (1991).

e - The oral RfDs for cadmium are 0.0005 mg/kg-d (water) and 0.001 mg/kg-d (food). The RfD of 0.0005 mg/kg-d is used for all oral exposures.

f - Recommended in the NYSDOH Ambient Air Criteria Document for this compound.

g - Based on the drinking water standard of 1.3 mg/L reported in HEAST (1993).

h - Extrapolated from the highest intermediate duration NOAEL listed in ATSDR (1990a) - for adverse respiratory effects in rabbits - by applying a safety factor of 1000.

i - Lead is a non-threshold neurotoxin. Reference doses and concentrations are thus inappropriate.

j - The oral RfDs for manganese are 0.0005 mg/kg-d (water) and 0.14 mg/kg-d (food). The RfD of 0.0005 mg/kg-d is used for all oral exposures.

k - Extrapolated from the oral reference dose assuming an inhalation rate of 20 m³/day and a body weight of 70 kg.

l - Extrapolated from the chronic less serious effect level reported in ATSDR (1990b) - for subjective complaints among exposed workers - by applying a safety factor of 10.

m - Extrapolated from a chronic NOAEL reported in ATSDR (1992e) - for absence of adverse cardiovascular and gastrointestinal effects in exposed workers - by applying a safety factor of 10.

n - Based on the Federal Standard for Particulate Matter (50 µg/m³) as discussed in NYSDOH (1988).

o - Based on the toxicity values for pyridine.

p - Extrapolated from the inhalation RAC by multiplying by 20 m³ per day inhalation rate and dividing by 70 kg body weight.

q - Extrapolated from a chronic NOAEL listed in ATSDR (1993g) - for absence of adverse hematologic effects in exposed workers - by applying a safety factor of 10.

r - Extrapolated from the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) converted to mg/m³, multiplied by the fraction of a week spent at work (8/24*5/7), and divided by a safety factor of 10 to account for intra human variation.

7 Characterization of chronic risks

This chapter presents the assessment of chronic risks to health that may ensue from operation of the NEPERA incinerator. Sections 7.1 and 7.2 present the quantitative estimates of risk for carcinogenic and non-carcinogenic end points derived from the multi-pathway risk assessment. Lead is assessed separately in Section 7.3 by means of calculations that relate lead concentrations in various media (air, soil, food, etc.) to changes in levels of lead in blood. Tables presenting the contaminant- and route-specific contributions to risk for each MEI are provided at the end of the chapter.

7.1 Estimates of carcinogenic risks to adults

Estimates of the incremental lifetime cancer risks that may ensue from operation of the NEPERA incinerator are calculated as the product of lifetime (that is, 70 years) exposure and carcinogenic potencies. A table summarizing the daily exposures to the adult MEI are provided at the end of Chapter 4, and carcinogenic potencies are given in Chapter 5. Risks are calculated for each exposure to a known or suspected carcinogen; the formulae used to calculate risks differ depending upon the mode of exposure. In the equations that follow, subscripts indicate that each risk estimate pertains to a particular combination of chemical and exposure pathway. The subscript "c" universally refers to chemical, while the subscripts "i", "o", and "dw" differentiate the inhalation, oral, and dermal-water pathways, respectively.

For inhalation exposure, incremental cancer risk ($R_{c,i}$) is estimated as the product of the time-averaged exposure point concentration in air ($c_{c,arc}$) and the unit risk factor (U_i) discussed in Chapter 6:

$$R_{c,i} = c_{c,arc} U_i \quad (7.1)$$

Risks from oral pathways ($R_{c,o}$) are estimated as the product of the oral potency slope (P_o) discussed in Chapter 6 and the estimated exposure (e_o):

$$R_{c,o} = P_o e_o \quad (7.2)$$

where e_o can be the estimated dose from any of the oral intake routes assessed in Chapter 5. Similarly, the incremental cancer risks $R_{c,dw}$ incurred from dermal exposures to water (e_{ds}) are estimated from the dermal potency factors (P_{dw}) derived in Chapter 6:

$$R_{c,dw} = P_{dw} e_{ds} \quad (7.3)$$

Since we assume, by convention, that the risk estimates are additive, a total estimate of the incremental risk of cancer (R_{total}) is provided by the sum of the individual estimates derived for each combination of chemical and exposure pathway:

$$R_{total} = \sum_{\text{chemicals}} \left(R_{c,i} + \sum_{\text{oral pathways}} \{ R_{c,o} \} + R_{c,dw} \right) \quad (7.4)$$

Estimates of R_{total} are compiled in Table 7.1 for the adult MEI. As shown, the estimated, incremental, upper-bound lifetime cancer risk for the adult MEI is seven in one million. Table 7.1 also gives a compilation of the total risk estimate by individual chemicals and exposure pathways. Blank entries indicate that either the contaminant is not a suspected or known human carcinogen, or that no estimate of carcinogenic potency is available. Sums of risk estimates are presented in the bottom and right margins (in the next-to-last line and next-to-last column, respectively). The risk estimate sums in the bottom margin represent totals for exposure pathways that are summed over all chemicals, while the risk estimate sums in the right margin represent totals for chemicals that are totaled over all exposure pathways. The total incremental cancer risk estimate is found at the intersection of the next-to-last line and the next-to-last column. In addition, fractional contributions of the total risk estimate are presented in the last line and last column of the table. Values in the last line indicate the fraction of the total risk estimate attributable to the pathway, while values in the right-most column denote the fraction of the total risk estimate due to each chemical. By definition, the values in the last line or the right-most column sum to unity (within the errors introduced by rounding).

The cancer risk estimates are presented with three significant figures in Table 7.1. Since these estimates are calculated in an electronic spreadsheet, seven or eight significant figures are available, consistent with the precision of the software. Regardless, it must be borne in mind that these estimates have an *accuracy* that corresponds to less than one significant digit, and even then, only as estimates of an upper bound on risk. Three significant figures are maintained only for the convenience of the reader who may wish to reproduce the calculations.

The estimate of increased lifetime cancer risk for the MEI is dominated by a single pathway of exposure and a single chemical. The most significant route of exposure is inhalation, which accounts for more than 99% of the total risk estimate. Hexavalent chromium is the principal chemical, contributing 90% of the total risk. Nickel and arsenic account for 7% and 1.4% of the total, respectively.

7.2 Estimates of non-carcinogenic risks to children

Estimates of the risks of adverse health effects other than cancer are assessed through the use of hazard ratios. As noted above, hazard ratios are calculated for the 2.5-year-old child MEI halfway through the assumed 70-year operational period of the plant. A table summarizing exposures to the child MEI is given in Chapter 4. A hazard ratio is defined generically as an estimated exposure concentration or dose divided by a reference concentration or dose that corresponds to a level at which no adverse health effects are anticipated. As for cancer risk estimates, the formulae for computing hazard ratios are differentiated by the mode of exposure. Inhalation hazard ratios ($H_{c,i}$) are defined as the ratio of the modeled concentration of a contaminant in air (c_{tox}) to its reference concentration (R_{IC}^i):

$$H_{c,i} = \frac{c_{tox}}{R_{IC}^i} \quad (7.5)$$

where the subscripts "c" and "i" are used again to signify that the hazard ratio is defined for a particular combination of chemical and exposure route (in this case, inhalation). Similarly, the hazard ratios for oral and dermal-water exposures are designated by the symbols $H_{c,o}$ and $H_{c,dw}$ respectively. These ratios are calculated by the ratio of exposure (e) to a reference dose. Using the nomenclature of Chapter 6, the respective definitions of hazard ratios are:

$$H_{c,o} = \frac{e_o}{R_{ID}^o} \quad (7.6)$$

$$H_{c,dw} = \frac{e_{ds}}{R_{ID}^{dw}}$$

The subscript "o" used to indicate oral exposures denotes many of the exposure routes evaluated in Chapter 5.

As a screening criterion, we define a total hazard index H_{total} to be the arithmetic sum of all of the individual hazard ratios over all chemicals and exposure routes.

$$H_{total} = \sum_{\text{chemicals}} \left(H_{c,i} + \sum_{\text{oral pathways}} \{ H_{c,o} \} + H_{c,dw} \right) \quad (7.7)$$

H_{total} for the MEI child equals 0.17. More detailed information on the contaminant- and route-specific contributions to the total hazard index is given in Table 7.2, in which columns correspond

to exposure pathways and rows correspond to individual chemicals. Sums of hazard ratios over each exposure pathway (and across all chemicals) are contained in the next-to-last line in the bottom margin of each table, and sums over each chemical (and across all pathways) are presented in the next-to-last column in the right margin. The intersection of the next-to-last line and next-to-last column, which denotes the sum over all chemicals and exposure routes, is the total hazard index (R_{total}).

Just as is the case for the estimate of total lifetime cancer risk, the estimate of hazard index for the child MEI is dominated by a minority of chemicals and exposure routes. The dominant exposure routes are inhalation (90%) and ingestion of fish (7%). The principal chemical contributor is nickel (61%).

7.3 Lead

Because the toxicity of lead to the developing nervous system may not exhibit a threshold — a dose below which no effect is likely — exposures to lead cannot be evaluated using reference doses or concentrations. Instead, one can use data describing the changes in the level of lead in the bloodstream that occur following an exposure to lead, and compare predictions to typical blood lead concentrations and concentrations associated with particular health effects. This section describes such an analysis, examining increments caused by exposures of child MEIs to lead in air, soil, water, and food. The magnitudes of coefficients, or slopes, relating environmental concentrations of lead to equilibrium blood lead increments have been extensively reviewed (U.S. EPA, 1986; U.S. EPA, 1990). Slopes, exposure point concentrations, and predicted increases in blood lead concentrations resulting from both maximum and anticipated emission rates are given in Table 7.3 for the child MEI.

For particulate lead at air concentrations below about $3 \mu\text{g}/\text{m}^3$, and for individuals with blood-lead concentrations below about $30 \mu\text{g}/\text{dl}$, studies on individuals (adults) yield coefficients in the range 0 to $3.6 \mu\text{g}/\text{dl}$ per $\mu\text{g}/\text{m}^3$. Population studies examining adults and children yield a median estimate of the coefficient as approximately $2 \mu\text{g}/\text{dl}$ per $\mu\text{g}/\text{m}^3$, which is the coefficient used here for children. The range of best estimates for population studies is approximately 1.3 to $2.5 \mu\text{g}/\text{dl}$ per $\mu\text{g}/\text{m}^3$, although the uncertainty ranges for these studies taken individually extend from approximately 1 to $4.4 \mu\text{g}/\text{dl}$ per $\mu\text{g}/\text{m}^3$. In the vicinity of the NEPERA incinerator, the average concentration of lead in air is expected to be similar to background values elsewhere in the U.S. — typically less than $0.1 \mu\text{g}/\text{m}^3$. The incinerator *per se*, at the worst case point, will add only $0.0004 \mu\text{g}/\text{m}^3$ — about 0.4% of the background value.

The coefficients relating blood lead concentration and air lead concentration address direct inhalation route only. Lead in particulates will deposit on soil and vegetation, and be ingested with soil and vegetation. Exposure to soil may be assessed either by examination of empirical

coefficients relating equilibrium blood lead concentrations to soil lead concentration, or by accounting for the amount of lead ingested with soil. This assessment uses the second approach, since it turns out to be more conservative.¹ The second approach is also that used to assess lead ingested with food and water.

The relation between equilibrium blood lead concentration and the rate of ingestion of lead is clearly non-linear at intake rates corresponding to relatively high blood lead levels ($>10 \mu\text{g/dl}$). The incremental effect on blood lead of equal increments in intake clearly decreases at high intakes (hundreds of $\mu\text{g/day}$ or more). At low intake rates (and low blood lead concentrations), the relation may be linear, but there is little confirmatory experimental evidence. The study giving the highest coefficient is that of Ryu *et al.* (1983), performed on infants less than 7 months old. U.S. EPA's analysis (U.S. EPA, 1986) of this study gives a coefficient of $0.16 \mu\text{g/dl per } \mu\text{g/day}$, although the children were not at steady state at the end of the experiment. All other studies of lead ingestion by food and water found substantially lower coefficients, but they were all performed at relatively high levels of lead intake. This assessment will use the value of $0.16 \mu\text{g/dl per } \mu\text{g/day}$, and apply it to all ingestion intakes (diet and water) for children.

This choice of coefficient for dietary exposures, and the other assumptions included in this assessment, ensure that the effects of soil lead are over-estimated compared with the use of a coefficient relating blood lead concentration with soil lead concentration. For example, with the assumptions used in this assessment, a 2.5-year-old child at the worst-case point will have an intake of approximately $4.7 \times 10^{-4} \mu\text{g/day}$ from soil ingestion, leading to an estimated increment in blood lead concentration of $7.4 \times 10^{-5} \mu\text{g/dl}$, greater than the highest end estimate for a correlation between blood lead concentration and soil lead concentration.

The final route of exposure to lead is dermal absorption during swimming. Since this assessment evaluates absorbed doses by this route, the coefficients obtained above for ingestion are underestimates — those coefficients relate exposure, not absorption, to blood lead concentration. Application of a typical gastrointestinal absorption factor of 50% for children yields a coefficient of approximately $0.4 \mu\text{g/dl per } \mu\text{g/day}$ for dermal absorption.

¹ The first method generates the following analysis. Various studies (summarized in U.S. EPA, 1986) have examined the relationship between blood lead concentration and soil (and dust) lead concentration. Coefficients between 6×10^{-4} and $7 \times 10^{-3} \mu\text{g/dl per ppm}$ in soil are found, with a reasonable median estimate of $2 \times 10^{-3} \mu\text{g/dl per ppm}$. The maximum soil lead concentration increment due to incinerator operations is approximately 0.007 ppm, so that use of such coefficients would generate estimates of the blood lead increment of $4 \times 10^{-6} \mu\text{g/dl}$ to $5 \times 10^{-5} \mu\text{g/dl}$. The higher value is still significantly lower than increment predicted by the second approach.

The increase in the concentration of lead in the blood of child MEI, using the slope factors just described, is about $2 \times 10^{-3} \mu\text{g/dl}$. This increase represents less than 0.06% of the average blood level ($4 \mu\text{g/dl}$) in U.S. children (ATSDR, 1993e). The Centers for Disease Control does not consider children with levels below $10 \mu\text{g/dl}$ to be poisoned (CDC, 1991). An increase of 0.06% would not be measurable and would not significantly alter a child's health.

The total daily intake of lead by an MEI child is approximately $0.014 \mu\text{g/day}$ (assuming total absorption of inhaled lead). This intake is 0.3% of the average intake of lead for 2-year-old male children in 1990 (ATSDR, 1993e), and represents an insignificant increase in lead exposure.

Table 7. Lifetime cancer risks for the adult MEI

Contaminant	Direct inhalation	Soil ingestion	Leafy produce	Exposed produce	Protected produce	Milk	Beef	Fish	Drinking water	Swimming dermal	Swimming oral	Total for chemical	Fraction of total
Ammonia	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Antimony	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Arsenic	7.52e-08	7.66e-11	2.59e-09	2.32e-09	2.22e-10	2.85e-10	9.20e-10	3.67e-09	5.14e-09	7.76e-12	2.38e-11	9.04e-08	0.01354
Barium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beryllium	2.30e-09	1.03e-11	3.28e-10	2.98e-10	7.50e-12	5.47e-13	5.92e-11	2.14e-10	6.94e-10	9.95e-11	3.21e-12	4.02e-09	0.00060
Cadmium	2.09e-08	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.09e-08	0.00314
Chromium VI	6.03e-06	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	6.03e-06	0.90298
Chromium (total)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Copper	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Manganese	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Mercury	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Nickel	4.83e-07	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	4.83e-07	0.07237
Selenium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Silver	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Thallium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Zinc	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Alkyl pyridine	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-Cyanopyridine	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3-Cyanopyridine	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	4.90e-08	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	4.90e-08	0.00734
Ethanol	1.70e-10	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	1.70e-10	0.00003
Isopropanol	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Methanol	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-Picoline	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3-Picoline	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pyridine	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Toluene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total from route	6.66e-06	8.70e-11	2.92e-09	2.62e-09	2.30e-10	2.85e-10	9.79e-10	3.88e-09	5.84e-09	1.07e-10	2.70e-11	6.68e-06	
Fraction of total	0.99746	0.00001	0.00044	0.00039	0.00003	0.00004	0.00015	0.00058	0.00087	0.00002	0.00000		

NA = Not applicable

Table 7. Hazard ratios for 2.5 year-old MEI to assess health endpoint. than cancer

Contaminant	Direct inhalation	Soil ingestion	Leafy produce	Exposed produce	Protected produce	Milk	Beef	Fish	Drinking water	Swimming dermal	Swimming oral	Total for chemical	Fraction of total
Ammonia	1.78e-04	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	1.78e-04	0.00108
Antimony	1.15e-05	4.92e-05	2.20e-04	1.85e-04	7.12e-05	9.53e-05	2.42e-05	3.50e-06	2.75e-04	7.36e-05	7.63e-06	1.02e-03	0.00614
Arsenic	3.50e-05	4.64e-06	1.55e-05	1.39e-05	1.34e-06	4.24e-06	3.65e-06	1.45e-05	2.60e-05	7.31e-08	7.20e-07	1.20e-04	0.00072
Barium	9.51e-04	5.41e-07	2.22e-06	1.78e-06	3.91e-07	3.41e-06	3.72e-08	3.85e-06	3.03e-06	1.62e-07	8.39e-08	9.66e-04	0.00584
Beryllium	9.60e-05	1.53e-08	4.77e-08	4.38e-08	1.11e-09	1.99e-10	5.72e-09	2.07e-08	8.56e-08	2.29e-08	2.37e-09	9.62e-05	0.00058
Cadmium	5.24e-04	1.67e-06	1.16e-05	1.25e-05	1.21e-05	4.85e-05	6.64e-07	9.62e-06	9.34e-06	4.99e-07	2.59e-07	6.30e-04	0.00381
Chromium VI	1.21e-03	1.92e-06	5.95e-06	5.68e-06	4.17e-07	4.16e-05	3.95e-06	2.19e-06	1.08e-05	1.44e-06	2.98e-07	1.28e-03	0.00773
Chromium (total)	7.60e-03	6.06e-08	1.88e-07	1.79e-07	1.31e-08	1.31e-06	1.24e-07	6.89e-08	3.39e-07	4.53e-08	9.39e-09	7.60e-03	0.04593
Copper	7.96e-04	1.03e-06	6.05e-06	1.09e-05	1.24e-05	4.07e-05	6.78e-06	1.46e-05	5.76e-06	2.56e-08	1.59e-07	8.95e-04	0.00541
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Manganese	5.08e-03	2.43e-05	1.17e-04	1.06e-04	5.86e-05	1.77e-04	5.11e-06	1.73e-04	1.36e-04	7.27e-06	3.77e-06	5.89e-03	0.03559
Mercury	3.63e-04	2.89e-05	2.72e-04	2.62e-04	2.79e-04	4.96e-04	4.07e-04	1.13e-02	1.62e-04	2.88e-06	4.48e-06	1.36e-02	0.08212
Nickel	1.01e-01	8.02e-06	2.78e-05	3.76e-05	2.32e-05	1.30e-04	2.01e-05	2.68e-05	4.49e-05	6.00e-07	1.24e-06	1.01e-01	0.61026
Selenium	5.57e-07	1.55e-07	5.01e-07	5.59e-07	1.87e-07	9.36e-06	9.05e-07	1.77e-07	8.70e-07	2.91e-09	2.41e-08	1.33e-05	0.00008
Silver	4.43e-06	2.82e-07	1.66e-06	1.68e-06	1.36e-06	1.41e-04	5.29e-07	5.63e-07	1.58e-06	2.01e-08	4.38e-08	1.53e-04	0.00092
Thallium	1.04e-06	2.27e-06	6.98e-06	6.42e-06	4.38e-08	6.50e-05	3.36e-05	2.31e-04	1.27e-05	3.40e-08	3.52e-07	3.59e-04	0.00217
Vanadium	1.34e-04	3.04e-07	9.39e-07	8.86e-07	4.40e-08	8.75e-06	2.83e-07	1.19e-04	1.70e-06	4.55e-07	4.72e-08	2.66e-04	0.00161
Zinc	5.34e-05	7.09e-07	9.69e-06	2.19e-05	3.08e-05	4.22e-04	1.03e-04	2.37e-06	3.97e-06	3.53e-08	1.10e-07	6.48e-04	0.00392
Alkyl pyridine	8.32e-03	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	8.32e-03	0.05028
2-Cyanopyridine	2.48e-03	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.48e-03	0.01496
3-Cyanopyridine	2.48e-03	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.48e-03	0.01496
Benzene	3.69e-05	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	3.69e-05	0.00022
Ethanol	1.02e-07	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	1.02e-07	6.2e-07
Isopropanol	2.00e-07	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.00e-07	1.2e-06
Methanol	2.63e-06	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.63e-06	1.6e-05
2-Picoline	2.11e-03	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.11e-03	0.01274
3-Picoline	2.11e-03	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.11e-03	0.01277
Pyridine	1.33e-02	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	1.33e-02	0.08013
Toluene	2.30e-06	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	2.30e-06	1.4e-05
Total for route	1.49e-01	1.24e-04	6.98e-04	6.67e-04	4.91e-04	1.68e-03	6.10e-04	1.19e-02	6.94e-04	8.72e-05	1.92e-05	1.66e-01	
Fraction of total	0.89734	0.00075	0.00422	0.00403	0.00297	0.01018	0.00369	0.07200	0.00419	0.00053	0.00012		

NA = Not applicable

Table 7.3 Increase in levels of blood lead in 2.5 year-old MEI

Route of exposure		Slope	Exposure rates	Blood lead ($\mu\text{g}/\text{dl}$)
Inhalation	Direct inhalation	2 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{m}^3$)	4.42e-04 $\mu\text{g}/\text{m}^3$	8.84e-04
Ingestion	Surface soil	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	4.65e-04 $\mu\text{g}/\text{day}$	7.44e-05
	Leafy produce	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	1.56e-03 $\mu\text{g}/\text{day}$	2.50e-04
	Exposed produce	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	1.44e-03 $\mu\text{g}/\text{day}$	2.30e-04
	Protected produce	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	2.02e-04 $\mu\text{g}/\text{day}$	3.23e-05
	Milk	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	1.79e-03 $\mu\text{g}/\text{day}$	2.86e-04
	Beef	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	5.53e-05 $\mu\text{g}/\text{day}$	8.84e-06
	Fish	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	1.62e-03 $\mu\text{g}/\text{day}$	2.59e-04
	Drinking water	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	2.60e-03 $\mu\text{g}/\text{day}$	4.16e-04
	Surface water while swimming	0.16 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	7.21e-05 $\mu\text{g}/\text{day}$	1.15e-05
Dermal	Dermal absorption while swimming	0.4 ($\mu\text{g}/\text{dl}$)/($\mu\text{g}/\text{day}$)	6.96e-06 $\mu\text{g}/\text{day}$	2.78e-06
Total blood lead				2.46e-03

8 References

- Anderson, E., Browne, N., Dulesky, S., Ramig, J., and Warn, T, *et al.* (1985). *Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments*. Washington, DC: U.S. EPA, Exposure Assessment Group.
- ATSDR (1989a). *Toxicological Profile for Selenium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1990a). *Toxicological Profile for Copper*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1990b). *Toxicological Profile for Silver*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992a). *Toxicological Profile for Antimony*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992b). *Toxicological Profile for Barium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992c). *Toxicological Profile for Manganese*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992d). *Toxicological Profile for Mercury*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992e). *Toxicological Profile for Thallium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992f). *Toxicological Profile for Vanadium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1992g). *Toxicological Profile for Zinc: Draft Update*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1993a). *Toxicological Profile for Arsenic*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1993b). *Toxicological Profile for Beryllium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.

- ATSDR (1993c). *Toxicological Profile for Cadmium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1993d). *Toxicological Profile for Chromium*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1993e). *Toxicological Profile for Lead*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1993f). *Toxicological Profile for Nickel*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- ATSDR (1993g). *Toxicological Profile for Benzene*. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- Baes, C.F., Sharp, R.D., Sjoreen, J., and Shore, R. (1984). *A Review and Analysis of the Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture*. Oak Ridge, TN: Oak Ridge National Laboratory (ORNL-5786).
- CDC (1991). *Preventing Lead Poisoning in Young Children — a Statement by the Centers for Disease Control — October 1991*. Center for Disease Control (CDC), U.S. Department of Health and Human Services.
- Diem, K. and Lentner, C. (1973). *Documenta Geigy Scientific Tables*. Ciba-Geigy, Ltd., Basle. (as cited in NYSDOH, 1991).
- Doll, R. and Peto, R. (1981). *The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today*. New York, NY: Oxford University Press.
- Four Nines, Inc. (1992). *Dispersion Modeling and Risk Assessment Protocol*, NEPERA, Inc., Harriman, NY.
- ENTROPY (1992). *Stationary Source Sampling Report: Reference No. 11179*. Prepared by ENTROPY Environmentalists Inc. for NEPERA, Inc., Harriman, NY. September, 1992.
- ENTROPY (1993a). *Stationary Source Sampling Report: Reference No. 11546*. Prepared by ENTROPY Environmentalists Inc. for NEPERA, Inc., Harriman, NY. February, 1993.
- ENTROPY (1993b). *Stationary Source Sampling Report: Reference No. 12455*. Prepared by ENTROPY Environmentalists Inc. for NEPERA, Inc., Harriman, NY. December, 1993.

- Haas, C.N. and Scheff, P.A. (1990). Estimation of Averages in Truncated Samples. *Environ. Sci. Technol.* 24:912-919.
- HEAST (1993). *Health Effects Assessment Summary Tables: Annual Update*. Washington, DC: Office of Research and Development, U.S. EPA. EPA 540-R-93-058.
- Hoffman, F.O. and Baes III, C.F. (1979). *A Statistical Analysis of Selected Parameters for Predicting Food Chain Transport and Internal Dose of Radionuclides*. Prepared for the U.S. Nuclear Regulatory Commission. NUREG/CR-1004
- Hoffman, J. (1994). Personal communication with Judy Hoffman, Manager of Public Affairs, NEPERA, Inc., Harriman, NY.
- Hull, L. (1994). Personal communication with Larry Hull, Cornell Cooperative Extension Office for Orange County, NY.
- IRIS (1994). Integrated Risk Information System. Supported by the U.S. Department of Health and Human Services and U.S. Environmental Protection Agency. Bethesda, MD: National Library of Medicine.
- Martin, S. (1994). Personal communication with Sam Martin, Manager of Engineering, NEPERA, Inc., Harriman, NY.
- McHale, S. (1994). Personal communication with S. Heather McHale, Process Engineer, Four Nines, Inc., Conshohocken, PA.
- Mills, W.B, Dean, J.D., Porcella, D.B., et al. (1982). *Water Quality Assessment: A Screening Procedure for Toxic and Conventional Contaminants: Parts 1, 2, and 3*. Athens, GA: U.S. EPA, Environmental Research Laboratory, Office of Research and Development. (EPA-600/6-82/004 a,b,c)
- NEPERA (1993). *Trial Burn Plan for the Incinerator at Nepera, Inc., Harriman, NY*. Revised February 1993.
- Northeast National Climate Center (1994). Personal communication.
- NYASS (1991). *New York Agricultural Statistics 1990-1991*. New York Agricultural Statistics Service (NYASS), New York State Department of Agriculture and Markets.
- NYASS (1993). *New York Agricultural Statistics 1992-1993*. New York Agricultural Statistics Service (NYASS), New York State Department of Agriculture and Markets.

- NYSDEC (1989). *New York State Air Guide-26. Guidelines for the Control of Toxic Ambient Air Contaminants*. Albany, NY: New York Department of Environmental Conservation.
- NYSDEC (1991). *Draft. New York State Air Guide-1. Guidelines for the Control of Toxic Ambient Air Contaminants*. Albany, NY: Division of Air Resources, New York Department of Environmental Conservation.
- NYSDOH (1988). *Ambient Air Criteria Document for Zinc*. Albany, NY: Bureau of Toxic Substance Assessment, New York State Department of Health.
- NYSDOH (1989a). *Ambient Air Criteria Document for Manganese*. Albany, NY: Bureau of Toxic Substance Assessment, New York State Department of Health.
- NYSDOH (1989b). *Ambient Air Criteria Document for Nickel*. Albany, NY: Bureau of Toxic Substance Assessment, New York State Department of Health.
- NYSDOH (1990a). *Ambient Air Criteria Document for Cadmium*. Albany, NY: Bureau of Toxic Substance Assessment, New York State Department of Health.
- NYSDOH (1990b). *Ambient Air Criteria Document for Chromium*. Albany, NY: Bureau of Toxic Substance Assessment, New York State Department of Health.
- NYSDOH (1990c). *Ambient Air Criteria Document for Vanadium*. Albany, NY: Bureau of Toxic Substance Assessment, New York State Department of Health.
- NYSDOH (1991). *Guidance for Exposure Assessment of Municipal Solid Waste and Hospital Waste and Hospital Waste Incinerator Emissions*. Albany, NY.
- Radike, M.J., Stemmer, K.L., and Bingham, E. (1981). Effect of ethanol on vinyl chloride carcinogenesis. *Env. Hlth. Persp.* 41:59-62.
- Ryu, J.E., Ziegler, E.E., Nelson, S.E., and Fomon, S.J. (1983). Dietary intake of lead and blood lead concentration in early infancy. *Am. J. Dis. Child.* 137:886-891. (as cited in U.S. EPA, 1986).
- Stuart, A. and Ord, J.K. (1987). *Kendall's Advanced Theory of Statistics, Volume 1: Distribution Theory*. New York: Oxford University Press.
- Suprenant, L. (1994). Personal communication with Leslie Suprenant, Fisheries Biologist, New York State Department of Environmental Conservation.

- Swanson, E. (1994). Personal communication with Eric Swanson, Rensselaer County Soil Conservation Service, U.S. Department of Agriculture.
- Taylor, E.W. (1958). *The Examination of Waters and Water Supplies*. Boston, MA: Little Brown & Company.
- USDA (1981). *Soil Survey of Orange County, New York*. U.S. Department of Agriculture, Soil Conservation Service.
- USDA (1983). *Food Intakes: Individuals in 48 states, Year 1977-78*. U.S. Department of Agriculture, Human Nutrition Information Service Report No. I-1. (as cited in NYSDOH, 1991).
- U.S. EPA (1985). *Health Assessment Document for Polychlorinated Dibenzo-p-Dioxins*. Office of Health and Environmental Assessment. EPA/600/8-84/014F. (as cited in ATSDR, 1989b).
- U.S. EPA (1986). *Air Quality Criteria for Lead*, Vols I-IV. EPA-600/8-83/028aF through 028dF, Environmental Criteria and Assessment Office, Research Triangle Park NC.
- U.S. EPA (1988). *Superfund Exposure Assessment Manual (SEAM)*. Washington, DC: Office of Remedial Response. EPA/5401-88/001.
- U.S. EPA (1989a). *Interim Final Risk Assessment Guidance for Superfund — Volume 1: Human Health Evaluation Manual*. Washington, DC: Office of Emergency and Remedial Response (OSWER Directive 9285.7-01a).
- U.S. EPA (1989b). *Exposure Factors Handbook*. Prepared by Versar, Inc. for the Office of Health and Environmental Assessment. EPA/600/8-89/043.
- U.S. EPA (1990). *Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation*. EPA-450/2-89-011, Office of Air Quality Planning and Standards, Research Triangle Park NC.
- U.S. EPA (1993a). Requirements for Preparation, Adoption, and Submittal of Implementation Plans (Final Rule – 40 CFR Part 51 *et al.*). *Federal Register* 58:137, July 20, 1993, pp. 38816–38884.
- U.S. EPA (1993b). *Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions: Review Draft*. Washington, DC: Office of Health and Environmental Assessment. EPA/600/AP-93/003

USGS (1981a). 7.5 minute topographic map for the Monroe Quadrangle. U.S. Geological Survey.

USGS (1981b). 7.5 minute topographic map for the Popolopen Lake Quadrangle. U.S. Geological Survey.

Vanoni, V.A. (1975). *Sedimentation Engineering*. New York: American Society of Civil Engineers.

Wischmeier, W.H., and Smith, D.D. (1978). Predicting rainfall erosion losses — A guide to conservation planning. *USDA Handbook No. 537*.

Yang, Y. and Nelson, C. (1986). An Estimation of Daily Food Usage Factors for Assessing Radionuclide Intakes in the U.S. Population. *Health Physics* 50:245-257. (as cited in NYSDOH, 1991).